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Particulate size-selective sampling is an important consideration in determining ambient air quality standards and threshold limit values for workplace exposures. Hazard evaluations, as well as risk analyses, can benefit from an improved understanding of factors affecting regional respiratory tract deposition of particles in man. Here, thoracic deposition and its component parts are examined, as a function of particulate size, for ventilation rates ranging from normal respiration to heavy exercise in individuals who are habitual mouth breathers and in those who normally employ oronasal breathing when minute ventilations exceed approximately 35 $Lmin^{-1}$. Nonlinear regression techniques were used to fit logistic models of the form $Y = [1 + e^{\alpha + \beta \log X}]^{-1}$ to data from tests measuring extrathoracic (ET) and tracheobronchial (TB) deposition. X was defined as an impaction parameter ($\rho d^2 Q$) and as the aerodynamic diameter of the particle (D_{ae}) for ET and TB deposition, respectively. The logistic models yielded significantly improved fits of the experimental data compared to previously used linear regression models. Our analyses demonstrate that the activity level of the exposed population should be taken into account to assess the potential health consequences from ambient or workplace exposures.

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INFLUENCE OF BREATHING MODE AND ACTIVITY LEVEL ON THE REGIONAL DEPOSITION OF INHALED PARTICLES AND IMPLICATIONS FOR REGULATORY STANDARDS

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Abstract Particulate size-selective sampling is an important consideration in determining ambient air quality standards and threshold limit values for workplace exposures. Hazard evaluations, as well as risk analyses, can benefit from an improved understanding of factors affecting regional respiratory tract deposition of particles in man. Here, thoracic deposition and its component parts are examined, as a function of particulate size, for ventilation rates ranging from normal respiration to heavy exercise in individuals who are habitual mouth breathers and in those who normally employ oronasal breathing when minute ventilations exceed approximately 35 $Lmin^{-1}$. Nonlinear regression techniques were used to fit logistic models of the form $Y = [1 + e^{\alpha + \beta \log X}]^{-1}$ to data from tests measuring extrathoracic (ET) and tracheobronchial (TB) deposition. X was defined as an impaction parameter ($\rho d^2 Q$) and as the aerodynamic diameter of the particle (D_{ae}) for ET and TB deposition, respectively. The logistic models yielded significantly improved fits of the experimental data compared to previously used linear regression models. Our analyses demonstrate that the activity level of the exposed population should be taken into account to assess the potential health consequences from ambient or workplace exposures.

Introduction Because the potential of atmospheric aerosols to adversely affect health is a function of mass median aerodynamic diameter (MMAD), hazard evaluation requires size-selective sampling of ambient contaminants. Therefore, determination of the size of particles penetrating oro- and nasopharyngeal airways that reach tracheobronchial (TB) and pulmonary (P) regions to pose a potential threat to human health is of importance. Over the past thirty years, there have been numerous experimental studies of total and regional respiratory tract deposition of insoluble particles in man, with major reviews by LIPPMANN (1977) and the EPA (1982). Both spontaneous and controlled breathing patterns have been used with airflow rates ranging from normal respiration to heavy exercise with either nasal or oral breathing. SWIFT and PROCTOR (1982) used the data of YU et al. (1981) and CHAN and LIPPMANN (1980) in analyses that addressed deposition with oronasal breathing. In the current study, published data from various

laboratories for extrathoracic (ET) and TB deposition, along with previously unpublished data of Lippmann and co-workers at New York University (NYU), were fit to logistic regression models prior to examining the influence of breathing mode and activity level on TB, P, and thoracic (TB + P) deposition.

Statistical Analysis Methods Using the digitized data of Fig. 1 in YU et al. (1981), the recent data of HEYDER (1984), and current NYU data, particulate losses in the ET region during nasal inspiration were examined with a logistic regression model of the form:

$$Y = [1 + e^{\alpha + \beta \log (\rho d^2 Q)}]^{-1} \qquad (1)$$

where ρ is the particle mass density (gcm^{-3}), d is the geometric particulate diameter (μm), and Q is the airflow rate ($cm^3 sec^{-1}$). The term $\rho d^2 Q$ defines an impaction parameter and has been found to be useful in characterizing the deposition of particles when inertial impaction is the dominant deposition mechanism. Nonlinear regression estimation techniques were used to fit Eq. (1). The use of a logistic model and nonlinear estimation has the advantage that no "breakpoint" for a series of linear regressions need be assumed. Also, the predicted deposition efficiency will asymptotically approach a lower bound of zero and an upper bound of one. Inspiratory mean deposition efficiency for mouth breathing was estimated in a similar manner using the digitized data of Fig. 3 from YU et al. (1981), the data of HEYDER (1984), and the new NYU data.

Regression analyses for TB deposition data, normalized to particles entering the trachea, utilized a logistic model based upon log D_{ae} rather than an impaction parameter to account for the fact that TB deposition is due primarily to inertial impaction and sedimentation in the upper and lower airways, respectively. By definition, D_{ae} is the diameter of a spherical particle of unit density (cgs system) having the same terminal

settling velocity as the particle under consideration (of arbitrary shape, size, density, etc.). To enable comparisons between controlled and spontaneous breathing patterns, separate regression analyses were performed on the TB data of LIPPMANN (1977), CHAN and LIPPMANN (1980), and HEYDER (1984). An additional analysis involved combining all of the data of these investigators with new data from NYU. The recent NYU experiments contain deposition data for particles with a $D_{ae} > 7$ μm, thereby adding new information on the deposition of larger particles. Unlike laboratory ET deposition data, the TB data are not evenly distributed across the range of particulate sizes of interest; thus, a weighted nonlinear regression was used. Weights that were a function of the reciprocal of the frequency of observations in a size interval allowed size intervals to contribute more uniformly to the overall regression without masking the dispersion present in the data within a given interval.

The data of NIINIMAA et al. (1981) were used to determine the proportionality of airflow between the nose and mouth as a function of activity level. Subjects who normally use oronasal breathing to augment respiratory airflow (normal augmenters) when minute ventilation ($\dot{V}_E$) exceeds about 35 $Lmin^{-1}$, have 45-53% of inhaled air pass nasally. In subjects who habitually breathe oronasally, hereafter referred to as mouth breathers, the nasal fraction of airflow is considerably less than that for normal augmenters at any given $\dot{V}_E$ (see Table 1).

Since the logistic curve was fit to TB deposition data expressed as a percentage of particles entering the trachea, the TB data were converted to absolute deposition fractions when thoracic deposition was examined. The P portion of thoracic deposition was based upon the model of YU (1978) for oral entry airflows and upon the curve of LIPPMANN (1977) for nasal entry airflows, corrected for the airflow proportionalities given in Table 1.

Results and Discussion After the ET data for nasal and oral inspiration contained in Figs. 1 and 3, respectively, of YU et al. (1981) were digitized, linear regressions were refit; the coefficients obtained were essentially the same as those proposed by the authors. The percentage of variation in the data explained by the linear model, R^2, was obtained during these reanalyses and found to be 68% for nasal inspiration and 46% for inspiration through the mouth. The logistic models yielded significantly improved fits of the ET experimental data in that the analogous overall R^2 values were 91% and 76%, respectively. Logistic model parameters for Eq. (1) for ET deposition were $\alpha = 7.163$ and $\beta = -1.968$ with nasal inspiration and $\alpha = 8.896$ and $\beta = -1.88$ with oral inspiration.

When separate logistic models based upon D_{ae} were fit to TB deposition data from investigators using spontaneous breathing (LIPPMANN, 1977; CHAN and LIPPMANN, 1980) and compared to a more controlled breathing pattern (HEYDER, 1984), the existence of reproducible differences related to the breathing pattern was evident. Greater than 95% of the variation in the data was explained in each of the investigator-specific logistic analyses. For small particles, spontaneous breathing yields a greater TB deposition of particles, while for larger particles the converse holds. When the digitized data were plotted, intersubject variability for TB deposition with spontaneous breathing was greater than the mean difference between the two breathing patterns. This mitigates concern about which breathing pattern is more appropriate for estimating deposition in man following exposure to ambient airborne contaminants, and a logistic model based upon the combined TB data of all investigators was used in all subsequent analyses. The combined TB data yielded a logistic model explaining approximately 96% of the variation in the data with model parameters being $\alpha = 3.493$, $\beta = -4.84$.

Using the ET and TB logistic model deposition curves and the data of Table 1 for the proportionality of airflow between the nose and mouth, we examined ET, TB, and TB + P deposition over a range of minute ventilations from resting respiration to heavy exercise for normal augmenters and mouth breathers. Minute ventilation curves for ET deposition are shown in Figure 1. For normal augmenters (Panel A, Fig. 1), deposition curves are distinct and shift to the right for larger values of ρd^2Q only when $\dot{V}_E$ exceeds the oronasal switching point (~ 35 $Lmin^{-1}$). However, for mouth breathers, there is an oral component of airflow at all minute ventilations (see Table 1) so that the ET deposition curves for this group are all distinct (Panel B, Fig. 1).

From the ET deposition values associated with the various particulate sizes used to calculate ρd^2Q, there is less deposition in the head at any given size of particle for mouth breathers compared to normal augmenters for a $\dot{V}_E$ of 10 $Lmin^{-1}$ (normal respiration). As $\dot{V}_E$ increases from 10 to 30 $Lmin^{-1}$, ET deposition increases for a given particulate size in normal augmenters. Deposition decreases at 40 $Lmin^{-1}$ due to switching to oronasal breathing before increasing again at higher $\dot{V}_E$ values. However, since mouth breathers have an oral component of airflow even at normal respiration, ET deposition is essentially a nondecreasing function at all particulate sizes.

Tracheobronchial and thoracic deposition curves are shown in Figures 2 and 3, respectively. For normal augmenters, the peaks of the TB deposition curves shift towards lower particulate sizes for $\dot{V}_E$ less than the oronasal switching point. Just beyond the oronasal switching point, deposition of larger particles greatly increases (Fig. 2, Panel A). In contrast, for mouth breathers, there is little variability in the particulate sizes associated with the peaks in the TB deposition curves, and TB deposition basically decreases as $\dot{V}_E$ increases (Fig. 2, Panel B).

In both normal augmenters and mouth breathers, thoracic deposition curves (Fig. 3) reflect the large contribution to P deposition of particles $\leq$ 5 μm D_{ae}. In normal augmenters at a minute ventilation of 40 $Lmin^{-1}$ (moderate activity involving oronasal breathing), TB deposition contributes approximately 86% of the thoracic deposition of 6 μm D_{ae} particles compared to only 55% in mouth breathers. However, for 3 μm D_{ae} particles, TB deposition comprises a similar amount (31-37%) of TB + P deposition in both groups. Independent of $\dot{V}_E$, P deposition is negligible for 12 μm D_{ae} or larger particles.

Thoracic deposition, as a function of $\dot{V}_E$, is less variable for mouth breathers compared to normal augmenters (Fig. 3), probably due to the greater oral component of airflow in mouth breathers at any given $\dot{V}_E$. As $\dot{V}_E$ increases, thoracic deposition for normal augmenters initially decreases for a given particulate size, increases through the oronasal switching point, and then decreases. For mouth breathers, however, there are minimal changes in deposition at lower ventilation rates with monotonic declines in deposition as $\dot{V}_E$ increases beyond 30 $Lmin^{-1}$.

From these and other results, it can be deduced that regional respiratory tract deposition of insoluble particles in man is a complex function of breathing route, ventilatory level, and particulate physical and aerodynamic properties. Our analyses demonstrate that the activity level of the exposed population should be taken into account to assess the potential health consequences from ambient or workplace exposures.

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Table 1. Proportionality of Airflow Between the Nose and Mouth as a Function of Minute Ventilation in Normal Augmenters and Mouth Breathers.†

		Minute Ventilation ($Lmin^{-1}$)				
		10	20	30	40	60
Normal Augmenters	Nose	1.00	1.00	1.00	.53	.45
	Mouth	.00	.00	.00	.47	.55
Mouth Breathers	Nose	.72	.47	.40	.34	.30
	Mouth	.30	.53	.60	.66	.70

†Data derived from Fig. 2 of NIINIMAA et. al. (1981).

Figure Legends

Figure 1. Extrathoracic deposition of particles for normal augmenters (Panel A) and mouth breathers (Panel B) as a function of an impaction parameter for minute ventilation, $\dot{V}_E$, ranging from a resting level (10 L min^{-1}) to heavy exercise (60 L min^{-1}).

Figure 2. Tracheobronchial deposition of particles for normal augmenters (Panel A) and mouth breathers (Panel B) as a function of aerodynamic particulate diameter for minute ventilation, $\dot{V}_E$, ranging from a resting level (10 L min^{-1}) to heavy exercise (60 L min^{-1}).

Figure 3. Thoracic deposition of particles for normal augmenters (Panel A) and mouth breathers (Panel B) as a function of aerodynamic particulate diameter for minute ventilation, $\dot{V}_E$, ranging from a resting level (10 L min^{-1}) to heavy exercise (60 L min^{-1}).

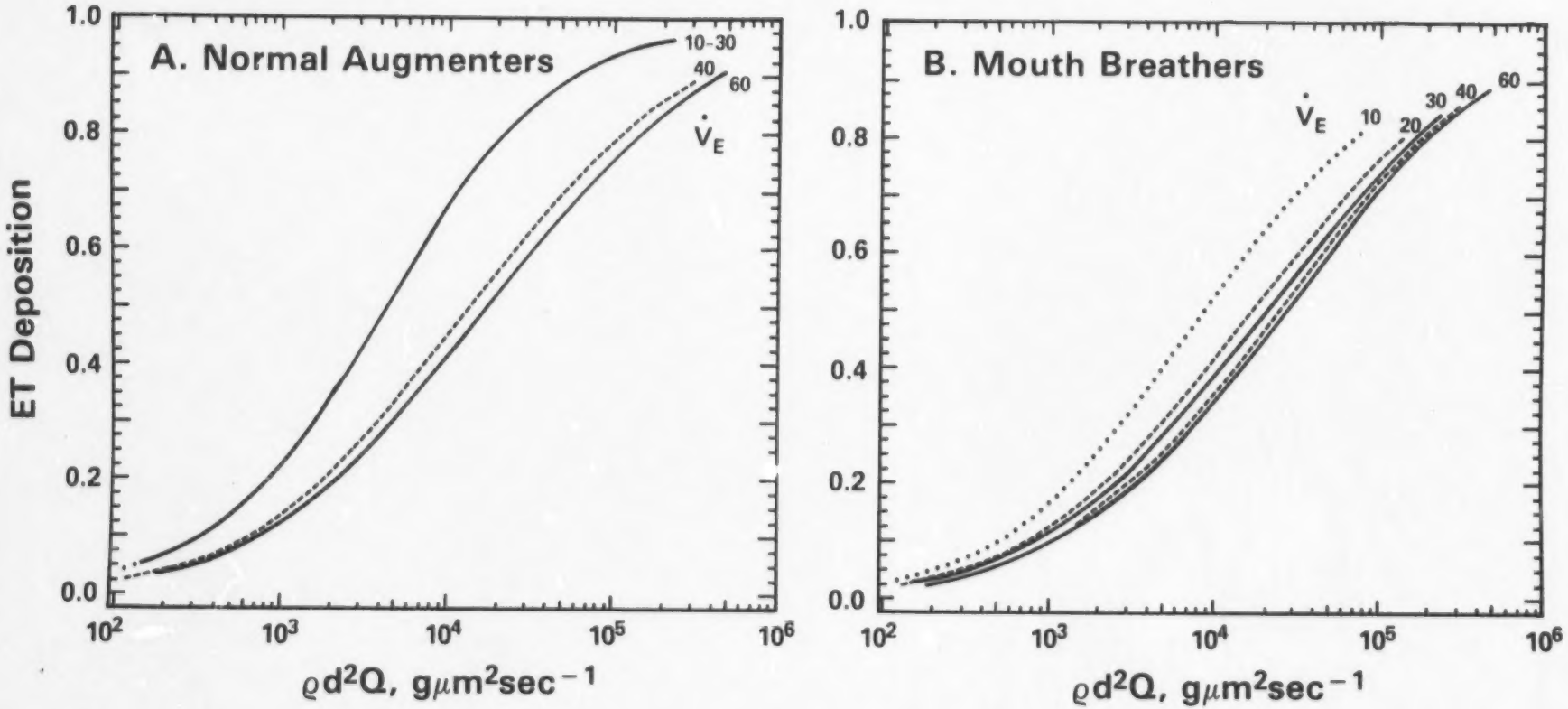
A. Normal Augmenters
B. Mouth Breathers
ET Deposition
1.0
0.8
0.6
0.4
0.2
0.0
10-30
40
60
$\dot{V}_E$
10
20
30
40
60
10^2
10^3
10^4
10^5
10^6
ϱd^2Q, $g\mu m^2 sec^{-1}$

Figure 1

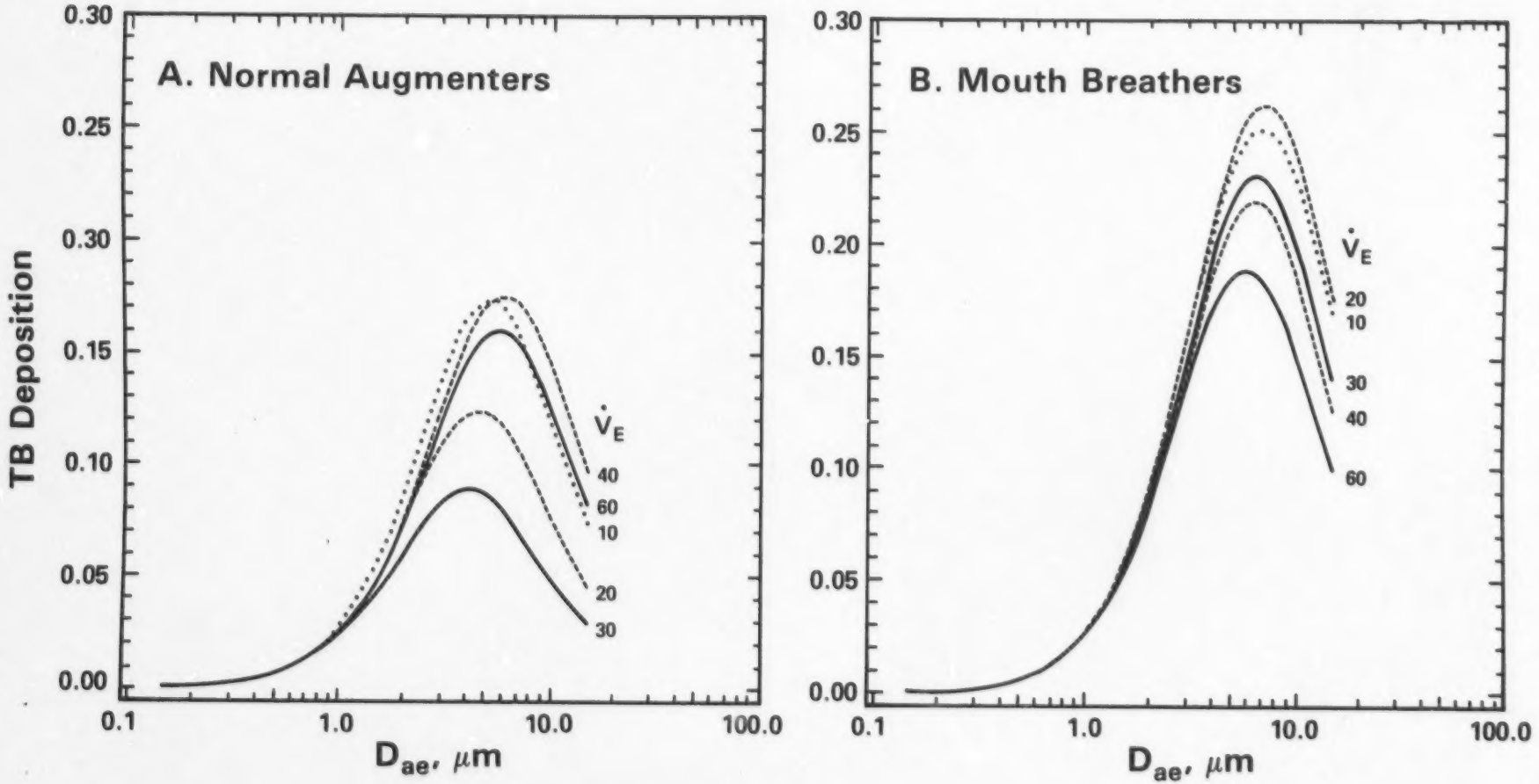
A. Normal Augmenters
B. Mouth Breathers
TB Deposition
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.1
1.0
10.0
100.0
D_{ae}, μm
$\dot{V}_E$
40
60
10
20
30

Figure 2

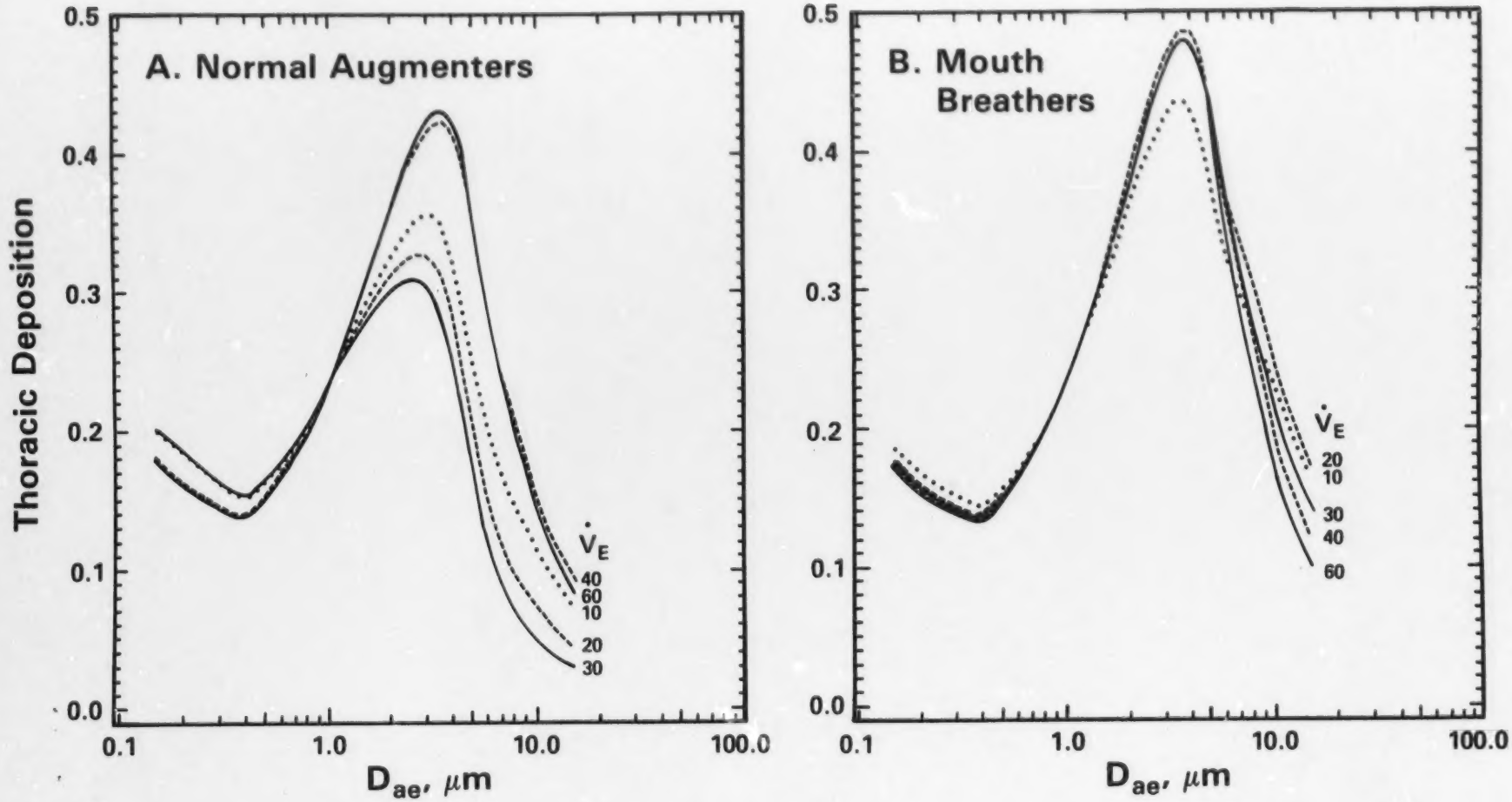
A. Normal Augmenters
B. Mouth Breathers
Thoracic Deposition
0.5
0.4
0.3
0.2
0.1
0.0
0.1
1.0
10.0
100.0
D_{ae}, μm
$\dot{V}_E$
40
60
10
20
30
20
10
30
40
60

Figure 3